

Racial Differences in Urban Children's Environmental Exposures to Lead

ABSTRACT

Objectives. This study explored whether differences in environmental lead exposures explain the racial disparity in children's blood lead levels.

Methods. Environmental sources of lead were identified for a random sample of 172 urban children.

Results. Blood lead levels were significantly higher among Black children. Lead-contamination of dust was higher in Black children's homes, and the condition of floors and interior paint was generally poorer. White children were more likely to put soil in their mouths and to suck their fingers, whereas Black children were more likely to put their mouths on window sills and to use a bottle. Major contributors to blood lead were interior lead exposures for Black children and exterior lead exposures for White children.

Conclusions. Differences in housing conditions and exposures to lead-contaminated house dust contribute strongly to the racial disparity in urban children's blood lead levels. (*Am J Public Health.* 1996;86:1460-1463)

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Introduction

In the United States, Black children are at increased risk for having an elevated blood lead level compared with White children.^{1,2} In the National Health and Nutrition Examination Surveys, Black children were found to have significantly higher blood lead levels than Whites, even after controlling for income and urban status.^{1,3} Other investigators have reported significant racial differences in blood lead levels among children in New York City and Massachusetts.^{2,4} However, these studies did not attempt to measure environmental exposures to lead. It therefore remained unknown whether differences in environmental exposures contribute to the racial disparity in children's blood lead levels.

Methods

Secondary analyses of data from a cross-sectional study, which has been described extensively elsewhere, were undertaken for children described by respondents as being either White or Black.⁵ Briefly, a random sample survey was used to identify and enroll 205 children, aged 12 to 31 months, who had resided in the same house in Rochester, NY, since at least 6 months of age. Children's blood and multiple measures of household dust, water, soil, and paint were analyzed for lead, and interviews were conducted with family members to ascertain the children's risk factors for having an elevated blood lead level. Enrollment, interviews, and sampling were conducted between August 29 and November 20, 1993.

Blood lead was obtained by venipuncture, and the detection limit for lead was set at 1.0 $\mu\text{g}/\text{dL}$, with a precision of $\pm 0.5 \mu\text{g}/\text{dL}$. To characterize children's exposure to lead-contaminated house dust (micrograms of lead per square foot), sampling was conducted by means of a wipe method. Dust lead loading was chosen as the unit of measure because it has been shown to be more predictive of children's blood lead levels than is dust lead concentration.⁵ Dust levels (grams

per square foot) were measured by using the Baltimore Repair and Maintenance (BRM) vacuum method.⁶ The lead content of interior paint was measured with an x-ray fluorescence analyzer (Microlead I, Warrington), and core soil samples were taken from the perimeter of the house's foundation, where bare soil was present. Respondents collected 1-minute flush water samples. Laboratory analyses and quality control performed on these samples have been extensively described elsewhere.⁵

For all statistical analyses, children's blood lead and serum ferritin levels and all environmental lead measurements were log transformed (base 10). Characteristics of Black children vs White children were tabulated and compared with *t* tests, the Wilcoxon test, chi-square tests, or Fisher's Exact Test. Multiple regression models were constructed to determine which covariates were significant predictors of children's blood lead after adjustment for other factors influencing blood lead. Variables included in the model selection process were those that were correlated with log(BPb) or, in the case of dichotomized variables, those that exhibited differences in the mean log(BPb) between the categories, provided there were at least four children in each category. These variables included dust lead loading, maximum interior paint concentration, condition of paint at site of maximum paint lead concentration, interaction of paint lead concentration and condition, water lead concentration, soil lead concentration, presence of bare soil, average frequency of household cleaning, child's serum ferritin level, child's ingestion of dirt or soil, time child spends outdoors, child's use of a bottle, race, household

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income, parent's college education, single-parent household, residence in rental housing, and age of house. The variables were investigated separately for Black children and White children, but the same variables were used for all models. All *P* values are two sided.

Results

Of the 205 children in the original study, 86 (42%) were Black, 86 (42%) were White, and the remaining 33 (16%) were described by the respondent as Hispanic or Puerto Rican (8%) or of other racial groups, including Asian, American Indian, and not given (8%). The geometric mean blood lead level for Black children was significantly higher than that for White children: 8.8 $\mu\text{g}/\text{dL}$ vs 4.7 $\mu\text{g}/\text{dL}$, respectively ($P < .001$). The socioeconomic status of Black children also was significantly different (Table 1).

Exposure to lead-contaminated house dust was significantly higher for Black children than for White ones ($P = .001$) (Table 2). In contrast, water lead levels were higher in White children's homes ($P = .029$). Soil lead levels were similar for both groups of children, but bare soil was present more often in the yards of White children ($P = .073$). Paint lead levels were not significantly different between groups, but there was a marginal difference in the condition of painted surfaces by child's race ($P = .092$). More Black children lived in homes with floors that were in poor condition ($P = .002$) and had higher dust levels ($P = .0001$).

Black children were more likely to mouth window sills ($P = .004$) and to use a bottle ($P = .022$), whereas White children were more likely to put soil in their mouths ($P = .043$) and to suck their thumbs or fingers ($P = .002$). Black children also were more likely to have been observed to eat paint chips. On average, White children were reported to spend 19 hours per week outdoors during the summer months, compared with 16 hours for Black children ($P = .227$).

A multivariate regression analysis including both Black and White children was developed to identify predictors of children's blood lead levels. This analysis indicated that Black race was the strongest predictor of blood lead (Table 3). To investigate why Black race remained an independent predictor after controlling for environmental exposures, two multiple regression models—one for Black children and a separate one for White

TABLE 1—Blood Lead and Serum Ferritin Levels, Demographic Status, Housing Characteristics, and Behaviors of 172 Urban Children, by Race

	Black (n = 86)	White (n = 86)	P
Child and family characteristics			
Mean age of child, mo (± 2 SD)	20.0 (8.8, 31.2)	21.2 (11.0, 31.4)	.15
Blood lead, ^a $\mu\text{g}/\text{dL}$ (± 2 SD)	8.8 (2.9, 26.8)	4.7 (1.8, 12.3)	<.001
Serum ferritin ^a (± 2 SD)	26.2 (7.6, 90.2)	22.8 (8.3, 62.9)	.11
Mean age of house, y (± 2 SD)	76.8 (35.8, 117.8)	70.0 (21.7, 118.3)	.049
Sex, % female	45	55	.22
Rental housing, %	93	27	<.001
Income < \$15 500, %	87	20	<.001
Single-parent household, %	77	15	<.001
Parent has less than high school education, %	88	28	<.001
Child's behaviors			
Chews on sill, %	37	17	.004
Uses a bottle, %	55	37	.022
Eats dirt or soil, %	20	34	.043
Sucks fingers, %	21	43	.002
Eats paint chips, %	13	5	.059
Time spent outside, ^b (mean ± 2 SD)	16 (−16, 48)	19 (−13, 51)	.227

^aGeometric mean.

^bHours spent outside on average each week.

TABLE 2—A Comparison of Urban Children's Environmental Exposures, Housing Conditions, and the Presence of Bare Soil, by Race

Source of Exposure	Black (n = 86)		White (n = 86)		P
	Geometric Mean	± 2 SD	Geometric Mean	± 2 SD	
Mean dust lead, $\mu\text{g}/\text{sq ft}$	135	10, 1825	73	11, 503	.0011
Mean dust, g/sq ft	.37	.06, 2.21	.16	0.03, 0.75	.0001
Water lead concentration, $\mu\text{g}/\text{L}$	7.4	1.2, 44.0	10.6	0.9, 127.1	.029
Soil lead concentration, $\mu\text{g}/\text{g}$	561	3, 96963	636	14, 29176	.715
Maximum paint lead concentration, mg/cm ²	6.4	0.3, 152.2	7.9	0.5, 123.2	.382
Condition of floors, % in poor condition	17		3		.002
Interior paint condition, ^a % poor or average	52		40		.092
Bare soil present, %	90		97		.073

^aPaint condition at the location of the maximum paint lead concentration.

children—were developed. For Black children, the sources of environmental lead exposures were generally from the interior environment: dust lead loading, condition and lead content of painted surfaces, and water lead concentration (Table 4). In contrast, sources of environmental lead exposures for White children were generally from the exterior environment: soil lead concentration, ingestion of dirt or soil, and amount of time spent outdoors.

Discussion

Racial disparity in urban children's blood lead levels appears to be due to differences in housing conditions and environmental exposures. While lead-contaminated soil and soil ingestion contribute to blood lead for both Black and White children, Black children, who in this study were largely impoverished and lived in poorly maintained rental housing, are also exposed to higher levels of

TABLE 3—Multivariate Regression Analysis^a: The Association of Blood Lead Levels among 172 Urban Children, by Environmental Lead Exposures, Demographic Characteristics, and Children's Behaviors

Variables	Parameter Estimate	Standard Error	P	R ²
Black race	.2267	.0413	.0001	11.5
Dust lead loading, µg/sq ft	.1603	.0339	.0001	8.6
Eats dirt or soil	.1514	.0378	.0001	6.1
Does not use bottle	.0784	.0338	.022	2.1
Water lead levels, µg/L	.0687	.0351	.052	1.5
Parent has high school education or less	.0732	.0423	.086	1.1

^aPercentage of variation explained (R²) is the independent contribution for each variable. The entire model explains 43% of the variation in children's blood lead levels. Blood lead, dust lead, and water lead levels have been log transformed.

TABLE 4—Multivariate Regression Analysis^a: The Association between Race and Blood Lead Levels among 172 Urban Children, by Environmental Lead Exposures, Demographic Characteristics, and Children's Behaviors

Variable	Parameter Estimate	Standard Error	P	R ²
Black children				
Dust lead loading	.3089	.0649	.0001	19.5
Paint lead variables			.007	11.7
Lead content of paint	.2188	.1854	.242	1.2
Paint condition	.1277	.0826	.127	2.1
Paint condition interaction ^a	-.1384	.0663	.041	3.8
Water lead levels	.1404	.0614	.026	4.5
Eats dirt or soil	.1137	.0614	.069	3.0
Soil lead variables			.080	4.4
Soil lead levels	.0685	.0429	.115	2.2
Soil present	-.0826	.1538	.593	0.2
White children				
Soil lead variables			.006	9.1
Soil lead levels	.1079	.0331	.002	9.1
Soil present	-.3251	.1446	.027	4.3
Time spent outdoors	.0039	.0013	.003	8.2
Eats dirt or soil	.1219	.0441	.007	6.5
Ferritin levels	.2199	.0918	.019	4.9
Single-parent household	.1147	.0548	.040	3.7

^aPercentage of variation explained (R²) is the independent contribution for each variable with a P value below .10. The entire model for Black children explains 44% of the variation in blood lead levels and the model for White children explains 32% of the variation. The variable for soil lead levels includes a term to indicate presence of bare soil. The combined effects of the three paint variables and two soil variables are shown on the lines "Paint/Soil Lead variables." Blood lead, dust lead, water lead, soil lead, and serum ferritin levels have been log transformed.

lead-contaminated house dust and to painted surfaces and floors that are in poorer condition. Thus, housing condition and exposure to lead-contaminated house dust appear to be major contributors to the racial disparity in children's blood lead levels.

Earlier studies have shown that lead-contaminated house dust is one of the

most important predictors of children's blood lead levels.^{5,7,8} In one study, which was reported in 1986 and predominantly included middle-class White children, lead-contaminated house dust was the strongest predictor of children's blood lead.⁸ Since then, however, lead deposition in house dust has declined, probably owing to the elimination of leaded gaso-

line. In the present analysis, exterior sources of lead were found to be the major sources of lead for White children, which is consistent with recent data showing that soil abatement is beneficial only for children who are not exposed to higher levels of lead-contaminated dust.⁹ In an earlier study of urban children (reported in 1980), soil ingestion was also found to contribute to White children's blood lead but was not a contributor for Black children.¹⁰ In the present study, Black children were less likely to ingest soil, but soil nevertheless contributed to their lead intake. Collectively, these data suggest that as lead contamination of house dust falls below a certain threshold, soil becomes a more important source of lead for urban children.

There are several possible reasons for this study's findings of higher levels of lead-contaminated house dust in Black children's homes. Painted surfaces in White children's homes generally had a higher lead content but were in better condition. Similarly, the floor conditions were better in White children's homes. Finally, although the types and frequency of cleaning differed by race, these differences did not alter the results of these analyses. Regardless of the specific reason for the differences in dust lead levels, these data indirectly support interim controls (i.e., temporary measures to reduce potential environmental exposures) as viable approaches to reducing children's exposure to lead.

The racial disparity in blood lead levels may also be explained, in part, by children's mouthing behaviors. White children were more likely to ingest soil and to suck their thumbs or fingers, whereas Black children were more likely to put their mouths on window sills and to use a bottle. Certain risk behaviors differed by children's race, but we were not able to determine whether these behaviors are culturally or environmentally driven. For example, if painted surfaces are in poor condition, paint chips are more likely to be accessible. Likewise, if paint is in poor condition, children may be more inclined to put their mouths on the window sills because lead-based paint is sweet. Nevertheless, with the exception of eating soil and not using a bottle, these behaviors did not appear to contribute to children's blood lead.

There are other potential reasons for the racial differences in blood lead levels. For example, persons with hemochromatosis or δ-aminolevulinic acid dehydratase isozyme 2 may have an increased rate of

lead absorption or retention, which could account for some of the racial differences in blood lead.^{11,12} Finally, there is a lower calcium intake among Black children, which has been shown to be inversely related to blood lead.^{13,14}

There are some limitations of these analyses. First, because we were not able to control for household income, our conclusions are limited to addressing why there is racial disparity in blood lead among urban children. Second, because we did not measure other potential modifiers of lead absorption, such as calcium intake, we were able to identify differences only in environmental exposures and behaviors and not in dietary intake or other biological aspects. Third, there is a delay between children's exposure to their environment and any effect of that exposure on their blood lead levels, and this delay may affect the estimate of this relationship. Finally, even though we used strict eligibility criteria, the cross-sectional design of this study may not adequately estimate prior exposure.

In conclusion, the racial disparity in blood lead levels among urban children appears to be largely due to differences in environmental exposures and housing conditions. Specifically, the homes of Black children had higher levels of lead-contaminated dust and their interior surfaces were in poorer condition. To a lesser extent, mouthing behaviors also appear to contribute to racial differences in blood lead. Although the benefit remains unproven, these data suggest that controlling lead-contaminated house dust

and improving the condition of housing will reduce the racial disparity in blood lead. □

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